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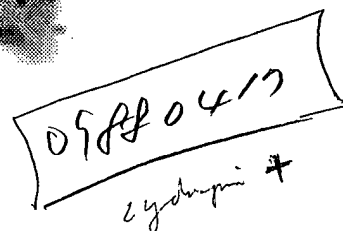
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http://www.google.com/search?q=cache:Mnd_HG6kVokC:www.taconic.com/anmodels/kotsgp53.htm+p53-/-+mice&hl=en&ie=UTF-8

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These search terms have been highlighted: **p53 mice**



TSG-p53[®] Knockout Mice **(Tumor Suppressor Gene Deficient)**

B6.129-*Trp53*^{tm1} N4 and B6.129-*Trp53*^{tm1} N5

Formerly C57BL/6Tac-*Trp53*^{tm1} N4 and C57BL/6Tac-*Trp53*^{tm1} N5

Order Model #'s:

P53N4-M (homozygotes)

P53N5-T (heterozygotes)

P53N5-W (wild type)

Coat Color: Black

Features: The TSG-p53 mice are deficient in the *Trp53* tumor suppressor gene. Recent studies indicate that the *Trp53* gene is the most commonly mutated gene in human cancers. This mouse can be used to study *Trp53* gene function, tumor biology or screen potential carcinogens.

Origin: Developed by Donehower and Bradley at the Baylor College of Medicine. Received at Taconic from the Bradley Lab in November 1991 at two backcrosses (N2) onto C57BL/6J from the (129/Sv x C57BL/6) chimera. Bred to N3 prior to cesarean derivation in December 1991 and to N4 immediately after derivation. Homozygote colony maintained at N4 through intercrossing of homozygous males and heterozygous females.

Heterozygote colony maintained at N5 through mating of N4 Homozygotes with C57BL/6NTac **mice**. Wild type control colony maintained at N5 through mating of N4 Wild Types with C57BL/6NTac **mice**.

Initial Publication: Donehower, L.A., Harvey, M., Slagle, B.L., McArthur, M.J., Montgomery, C.A. Jr., Butel, J.S., Bradley, A. (1992) **Mice** deficient for **p53** are developmentally normal but susceptible to spontaneous tumors, Nature, Vol. 356, pp. 215-221.

Updates:

New Applications:

Chromosomal Recombination in Tumor Development

Transgenic Animals for Carcinogenicity Testing

New Results:

Short-term Carcinogenicity Test

Redfield Laboratories Performing Carcinogenicity Studies

New Data: P-cresidine Positive Control

Links to Related Sites:

NIEHS Use of **p53 Mice** for Rapid Identification of Potential Carcinogens

Alternatives to Carcinogenicity Testing - ILSI/HESI Committee

Reference Library:

Use of Transgenic Models in Proposed Carcinogenicity Bioassay

Spontaneous Proliferative Lesions, Organ Weights, Body Weights

Health Reports by Health Report Group:

MBU 3A

Related Animal Models:

C57BL/6 TSG-**p53** N12 Knockout **Mice**

TSG-**p53**[®]/Big Blue[®] Knockout/Transgenic **Mice**

TG.AC Transgenic **Mice**

XPC Knockout **Mice**

pim-1 Microinjected **Mice**

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- The Models will be used for research purposes only.
- The Models will not be bred except to obtain embryos or fetuses required for research purposes unless the purchaser maintains a Research Crossbreeding Agreement with Taconic Farms, Inc.
- The Models and biological materials derived from them will not be distributed to third parties or used for commercial purposes.

Taconic Transgenic Models are produced in Isolated Barrier Unit (IBU™) facilities under MPF™ conditions, and shipped in Taconic Transit Cages (TTC™).



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<http://www.google.com/search?q=cache:9QKconHaCooC:www.ncbi.nlm.nih.gov/disease/p53.html+p53+gene+cancer&hl=en&ie=UTF-8>

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These search terms have been highlighted: **p53 gene cancer**

NCBI

Genes and disease

Map

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 X Y

NEW Genome View

p53 gene

on chromosome 17

Databases

PubMed
the literature

LocusLink
collection of
gene-related
information

Protein structure
molecule in 3D

OMIM
catalog of human
genes and disorders

Information

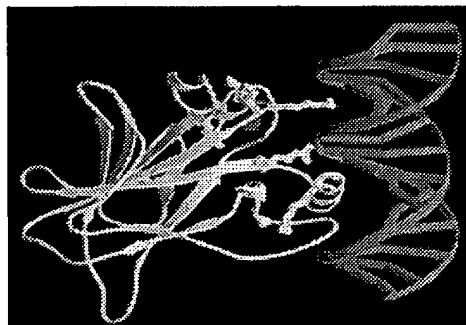
CancerNet
from the National
Cancer Institute, NIH

THE p53 GENE like the Rb **gene**, is a tumor suppressor **gene**, i.e., its activity stops the formation of tumors. If a person inherits only one functional copy of the **p53 gene** from their parents, they are predisposed to **cancer** and usually develop several independent tumors in a variety of tissues in early

adulthood. This condition is rare, and is known as Li-Fraumeni syndrome. However, mutations in **p53** are found in most tumor types, and so contribute to the complex network of molecular events leading to tumor formation.

The **p53 gene** has been mapped to chromosome 17. In the cell, **p53** protein binds DNA, which in turn stimulates another **gene** to produce a protein called p21 that interacts with a cell division-stimulating protein (cdk2). When p21 is complexed with cdk2 the cell cannot pass through to the next stage of cell division. Mutant **p53** can no longer bind DNA in an effective way, and as a consequence the p21 protein is not made available to act as the 'stop signal' for cell division. Thus cells divide uncontrollably, and form tumors.

Help with unraveling the molecular mechanisms of cancerous growth has come from the use of mice as models



The structure of the core domain of the p53 protein (light blue) bound to DNA (dark blue). The six most frequently mutated amino acids in human cancers are shown in yellow - all are residues important for p53 binding to DNA. Red ball: zinc atom. [Reproduced from Cho, Y., et al. (1994) Science, 265, 346-355, with kind permission.]

American Cancer
Society
research and patient
support

Oncolink
comprehensive
cancer information
from the University of
Pennsylvania

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cancerous growth has come from the use of mice as models for human **cancer**, in which powerful '**gene** knockout' techniques can be used. The amount of information that exists on all aspects of **p53** normal function and mutant expression in human cancers is now vast, reflecting its key role in the pathogenesis of human cancers. It is clear that **p53** is just one component of a network of events that culminate in tumor formation.

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<http://www.google.com/search?q=cache:fgH1RgdxmNQC:www.bioinfo.com/nihnov95a.html+cell+type+p53+gene+patent+cancer&hl=en&ie=UTF-8>

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These search terms have been highlighted: **cell type p53 gene patent cancer**

Federal Register Announcement, NIH, Late November 1995

Text of a *Federal Register* notice released by the Office of Technology Transfer, NIH, on November 24, 1995.

DEPARTMENT OF HEALTH AND HUMAN SERVICES
National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health

ACTION: Notice

The inventions listed below are owned by agencies of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign **patent** applications are filed on selected inventions to extend market coverage for U.S. companies and may also be available for licensing.

ADDRESS: Licensing information and copies of the U.S. **patent** applications and issued patents listed below may be obtained by writing to John Fahner-Vihtelic, Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Box 13, Rockville, Maryland 20852-3804 (telephone 301/496-7735 ext 285; fax 301/402-0220). A signed Confidential Disclosure Agreement (CDA) will be required to receive copies of the **patent** applications. Requests for copies of issued patents do not require the execution of a CDA.

Methods For Determining The Presence Of Functional **p53** In
Mammalian Cells
Fornace, A.J., Kastan, M.B. (NCI)

Filed 10 Aug 94

Serial No. 08/288,872 (CON of 07/974,960)

The protein **p53** is involved in tumorigenesis. Recent observations have indicated that the **gene** encoding **p53** is a tumor suppressor **gene**; however, mutation or deletion of this **gene** results in loss of this suppressor function. Mutations of the **p53 gene** have been demonstrated in tumors of the colon, breast, lung, ovary, bladder, and other organs, making the **p53 gene** the most commonly mutated **gene** yet identified in human cancers. While currently used assays can detect the presence of wild-type or mutant **p53** protein in mammalian cells, they cannot accurately determine the presence of functional **p53** protein in these cells, which is necessary to determine the biological function of functional **p53** and to develop subsequent diagnostic modalities using functional **p53**. This invention describes a specific **gene** whose expression is dependent on the presence of functional **p53** in cells and tumors, as well as methods by which the presence of this **gene** may be detected. It also describes a diagnostic kit utilizing a nucleic acid sequence capable of binding functional **p53**, which is then measured to detect **p53** presence. Issuance of a **patent** on this invention is currently pending. [portfolio: **Cancer** - Diagnostics]

Novel B-Lymphoma **Cell** Line And Antigen

Bock, G.H., Nelson, D.L., Kurman, C.C., Fleisher, T.A. (NCI)

Filed 9 Aug 94

Serial No. 08/287,718 (FWC of 07/934,106)

Various **cell** lines of B-**cell** lineage have been produced, but none have been of tumor **cell** origin. This case provides an IL-6 dependent B-**cell** lymphoma **cell** line, designated DS-1. The invention further provides a monoclonal antibody which reacts with the **cell** line and a method for detecting the presence of neoplastic cells by detecting the presence of an antigen on a **cell** which is not normal for that **cell type**. [portfolio: **Cancer** - Diagnostics; **Cancer** - Research Reagents]

Novel Human ras-Related Oncogenes Unmasked By Expression cDNA Cloning

Aaronson, S., Chan, A., Miki, T. (NCI)

Filed 24 May 94

Serial No. 08/247,946

A family of small G-proteins encoded by H-, K-, and N-ras is frequently activated as oncogenes in a wide variety of human tumors. Activation is usually due to a point mutation within the coding sequence which results in the molecule to be constitutively in the GTP bound (active) state. In normal cells, these proteins are coupled to growth factor signaling pathways and appear to cause proliferation or differentiation. Over the past several years, cloning efforts by many laboratories have

greatly expanded the number of ras-related proteins, to include R-ras, K-rev-1/rap and TC21. The present invention relates to a mutant TC21 protein that was cloned from an expression cDNA from a ovarian carcinoma **cell** line. Based upon the finding that an oncogenic form of TC21 exists, the present invention also relates to the generation of point mutations in R-ras for expression study. The present invention also relates to methods of diagnosing cancers or monitoring disease progression by detecting mutant forms of R-ras or TC21 at the protein or **gene** level. [portfolio: **Cancer** - Diagnostics; **Cancer** - Research Reagents]

Immortalized Adult Human Prostate Epithelial **Cell** Lines

Rhim, J.S., Webber, M.M. (NCI)

Filed 28 Apr 94

Serial No. 08/234,843

This invention relates to **cell** lines which are useful in testing compounds for anti-carcinogenic, anti-neoplastic, anti-invasive, or anti-metastatic activity by growing the **cell** line in the presence of the subject compounds. The **cell** lines contain DNA of a human Papilloma virus (HPV), either alone or with an activated viral ras oncogene, e.g., v-Ki-ras. The HPV immortalized line is not tumorigenic; however, the V-Ki-ras transformed HPV **cell** line is tumorigenic. They are useful for determining causes, treatment, and prevention of prostate **cancer**, benign prostate hyperplastic, male infertility, birth defects, aging, and assessment of environmental toxic agents. [portfolio: **Cancer** - Research Reagents]

Phosphonoalkyl Phenylalanine Compounds Suitably Protected For Use In Peptide Synthesis

Burke, T.R., Smyth, M.S., Lim, B.B. (NCI)

Filed 8 Jun 93

Serial No. 08/073,088

A novel class of phosphonodifluoromethyl phenylalanine ("F2Pmp") derivatives have been developed which are suitable for the synthesis of peptides containing the phosphotyrosyl (pTyr) mimetic, F2Pmp. These analogues bear Boc or Fmoc protection at the N α -position for either solution or solid-phase peptide synthesis using standard techniques. A number of studies have shown that peptides containing the F2Pmp residue show utility as inhibitors of src homology 2 (SH2) domain binding interactions and of phosphotyrosyl phosphatases. Unlike pTyr residues, the F2Pmp moiety is stable to both chemical and phosphatase-mediated hydrolysis, making it an attractive replacement for pTyr in signal transduction peptides. [portfolio: **Cancer** - Research Reagents]

Monoclonal Antibodies To Prostate Cells

Pastan, I. (NCI)

Filed 8 Oct 92

Serial No. 07/958,140

Monoclonal antibodies which bind to an antigen associated with prostate cells, including prostate **cancer**, can be used either individually or conjugated to drugs, labels, radioisotopes, or cytotoxins to target delivery of the conjugated to prostate cells. The antibodies are thus useful in a variety of diagnostic and therapeutic applications involving prostate **cancer**. A hybridoma **cell** line secreting monoclonal antibody PR1 is also provided, as well as methods for screening for the presence of metastatic prostate **cancer**. [portfolio: **Cancer** - Therapeutics]

Antibodies To Human LINE-1 p40 Protein

Fanning, T.G. (NCI)

Serial No. 07/750,044

Patent Issued 18 Jan 94

U.S. **Patent** No. 5,280,108

Antibodies to the human LINE-1 retrotransposon offer a powerful new tool for studying tumors. In most **cell** lines and tissues, human LINE-1 sequences (LIHs) are not expressed; however, LIH-specific RNA and proteins have been detected in **cell** lines and tissues derived from human germ **cell** tumors (teratocarcinomas) and breast tumors. These LIH antibodies, which are specific for the p40 protein portion of the retrotransposon, can be used for determining LIH expression in tumor cells and determining the role this retrotransposon plays in these cells. [portfolio: **Cancer** - Research Reagents]

Cartilage-Derived Morphogenetic Proteins

Luyten, F.P., Moos, M. Chang, S. (NIDR)

PCT Application PCT/US94/12814 filed 7 Nov 94

DHHS Reference No.: E-138-94/0

The present invention provides a cartilage-derived extract which initiates and promotes ectopic cartilage and bone development in vivo and recombinant cartilage-derived morphogenetic proteins which promote development of musculoskeletal tissues in vivo. These products will be useful in the therapeutic induction, repair, and maintenance of skeletal tissues. These compounds show promise for the healing of joint surface lesions and repair or reconstruction of cartilaginous tissues. They are also useful as growth factors for cells of the chondrocyte lineage which, expanded ex vivo, can be implanted into an individual where cartilage growth is desired. In addition, cloned polynucleotides encoding these proteins will be effective diagnostic reagents for detecting genetic abnormalities associated with poor skeletal development. [portfolio: **Cancer** - Therapeutics, biological response modifiers, growth factors]

Pulsed Low Frequency EPR Spectrometer And Imager

Bourg, J., Cherukuri, M., Mitchell, J., Mirotznik, M., Roth, B.,

Subramanian, S. (NCI) *

Serial No. 08/097,811

Patent Issued 7 Feb 95

U.S. **Patent** No. 5,387,867

This application describes an Electron Paramagnetic Resonance (EPR) spectroscopy imaging system. This system generates broadband pulses having a RF carrier frequency that is not highly absorbed by biological materials. The pulse generating system includes up and down chirp converters for frequency modulating of a carrier frequency and compression of the frequency modulated pulse to form a broadband excitation pulse of high energy. This technology's function has been proven and could form the basis of a clinical imaging device capable of high sensitivity to free radical species in human patients.
[portfolio: Devices/Instrumentation - Diagnostics, electron paramagnetic resonance]

***Federal Register* Announcement, NIH, Late November 1995**

Text of a *Federal Register* notice released by the Office of Technology Transfer, NIH, on November 24, 1995.

DEPARTMENT OF HEALTH AND HUMAN SERVICES
National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health

ACTION: Notice

SUMMARY: The inventions listed below are owned by an agency of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 or pursuant to 42 U.S.C. 241 to achieve expeditious commercialization of results of federally-funded research and development.

ADDRESS: Licensing information for the technologies referenced below may be obtained by contacting Stephen Finley, Ph.D., at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852-3804 (telephone 301/496-7056 ext 215; fax 301/402-0220).

cDNA Sequence of a Clone Encoding Arylalkylamine
N-acetyltransferase

Klein et al. (NICHD)

DHHS Reference No. E-161-95/0 and

Human **Gene** Encoding Serotonin N-acetyltransferase

Klein et al. (NICHD)

DHHS Reference No. E-222-95/0

The identification of an arylalkylamine N-acetyltransferase (AA-NAT) mRNA in the brain and the cloning of ovine and human cDNAs encoding for the pineal enzyme serotonin

N-acetyltransferase. These findings open a new area of research the importance of AA-NAT in the regulation of brain serotonin and the development of drugs which raise serotonin levels by inhibiting this enzyme. This enzyme is the rate-controlling step in the conversion of serotonin to melatonin.

The hormone melatonin has been linked to controlling circadian rhythms. Development of regulators of the synthesis of the hormone melatonin may be the preferred route to controlling seasonal reproduction cycles or sleep cycles of vertebrates.

Activators of the serotonin N-acetyltransferase may be beneficial to induce or enhance the quality of sleep at night. Inhibitors of serotonin N-acetyltransferase may lead to drugs that stimulate the levels of alertness and physical activity or delay the onset of fatigue. Licenses for the cDNAs encoding for this enzyme or the production of the enzyme are available.
